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THE OCULAR COMPLICATIONS OF TYPHOID FEVER.

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There are no ocular lesions characteristic of typhoid fever in the sense, for instance, in which certain forms of retinitis are pathognomonic symptoms of some varieties of so-called Bright's disease. Nevertheless, a number of eye-diseases may accompany or follow typhoid fever, precisely as they may be part of the symptomatology or sequels of other febrile affections which greatly depress or alter the nutrition of the patient. These ocular disorders have been recognized from the beginning of the last century, although necessarily in the earlier observations it is next to impossible to distinguish between typhus and typhoid. In recent times four systematic publications on this subject, in which the literature is fully reviewed, have appeared, namely, the articles of Knies,[1] Charles Stedman Bull,[2] Alvin A. Hubbell,[3] and my own chapter contributed to Professor Keen's monograph on "The Surgical Complications and Sequels of Typhoid Fever," published in 1898.

For the convenience of study I have ventured to suggest the following classification, arranged according to their probable relative frequency,[4] of the ocular complications and sequels of typhoid fever: (1) Affections of the conjunctiva and cornea; (2) affections of the retina and retinal vessels; (3) affections of the uveal tract—iris, ciliary body, choroid—and of the vitreous humor; (4) affections of the crystalline lens; (5) affections of the extra- and intraocular muscles; (6) affections of the optic nerve; (7) affections of the orbit and orbital circulation.

Ordinary conjunctivitis of the so-called catarrhal type is of frequent occurrence. So far as I am aware, bacteriological examinations are lacking in *typhoid-fever conjunctivitis*, but even if they had been made it is

doubtful whether they would have revealed any micro-organisms except those which are commonly present in the milder types of conjunctivitis and which probably have no specific relation to their development. During convalescence, and sometimes in the later stage of the disease, a true phlyctenular conjunctivitis appears, associated probably with more or less rhinitis. A phlyctenule may break down and form an ordinary *phlyctenular ulcer*, or without this origin *ulcerative keratitis* may arise, and if unchecked may develop into severe sloughing ulcers with hypopyon and secondary iritis, and may be followed by entire destruction of the eye.

The pathogenesis of these purulent ulcers may be twofold : (1) An infection may be carried through the blood-streams to the eye, that is, there is an autoinfection ; or, as Hubbell suggests, (2) the physical depression of the patient may be so great that the resisting power of the cornea, this structure being slightly anesthetic, is lessened, and some small abrasion of the epithelium permits an inoculation of pathogenic germs. In bad cases of typhoid fever, when the patient has lain for days in a comatose condition, with half closed eyelids, rapid melting down of the cornea, or *keratomalacia*, may occur, precisely as this takes place in cholera, dysentery and meningitis.

Fortunately, sloughing keratitis is a rare phenomenon in this disease (it appeared only twice in two thousand cases recorded by Hoelscher) ; but its prognosis is bad, especially when the general condition and surroundings of the patient are poor. Prompt treatment, however, may prevent disastrous consequences, as in a case reported by Veasey.

The treatment of the conjunctivitis of typhoid fever is that which is suited to any mild type of catarrhal inflammation of the conjunctiva—a collyrium of boric acid or common salt; washing the edges of the closed lids with tepid water and castile soap, and if there is free secretion of muco-pus, eversion of the lids and the application of silver nitrate (5 grains to 1 fluidounce), or of a 10% solution of protargol. Sloughing corneal ulcers call for atropin drops (4 grains to 1 fluidounce), with sufficient frequency to maintain mydriasis and

control iritis; frequent cleansing of the conjunctival sac with an antiseptic collyrium, boric acid or mercuric chlorid, 1·8000; direct applications to the ulcer after the sloughing-material has been cureted away of a 2% solution of silver nitrate, or pure carbolic acid, or tincture of iodin, or in very stubborn cases the actual cautery. In the intervals of treatment the ulcer should be covered with powdered iodoform. Hypopyon and corneal abscess indicate the usual surgical measures—incision, paracentesis, or Saemisch's section.

Infectious-disease iritis, or *irido-cyclitis*, which is not uncommon in relapsing, and typhus fever, is much rarer in typhoid fever, one case only having been reported by Sorel among 871 fever patients.

Choroiditis with vitreous opacities may occur, and there is some evidence to show that spontaneous inflammation of the vitreous, either in the form of flake-like opacities or of purulent deposits, may appear during the exhausting stages of the disease. Very rarely a *purulent choroiditis*, like that which is seen in pyemia, puerperal sepsis, or endocarditis, may complicate typhoid fever and result in phthisis bulbi, or shrinking of the ball. A good example of such an affection appearing in the third week of the disease is reported by Dr. C. W. Hall, of Kewanee, Ill.,[5] and another by Dr. B. L. Millikin[6] which began in the seventh or eighth week of her fever, and after two relapses. Unfortunately neither of these eyes were examined for microorganisms.

If suppuration in the vitreous or choroid has once begun, no treatment is likely to save the eye from destruction, intraocular injections of various antiseptic substances having proven to be of little value, with the single exception, perhaps, of chlorine water. But, as Hansell has suggested, if flakes of opacity are discovered early in the vitreous of a typhoid-fever patient, it is conceivable that vigorous supporting treatment may prevent the dreaded suppuration and the destruction of the eye. The presence of iritis indicates atropin, or similar mydriatic, in sufficient frequency to maintain mydriasis.

There seems little doubt that a certain number of *cataracts* develop as the result of typhoid fever (Trelat, Fontan, Romiée). These lenticular opacities (which

most frequently have been of the punctate variety) have been attributed by Fontan to a mechanical obstruction of the circulation, and by Romiée to an increase in the density of the serum and changes in the relation of the lens to the aqueous humor, but it seems to me more rational to believe that they depend upon an antecedent inflammation of the uveal tract, or at least upon a disturbance of its nutritive powers.

According to Charles Stedman Bull, *retinal hemorrhages* are not infrequent during the height of typhoid fever, being most common about the third week. They may appear in association with intestinal hemorrhage. I also have expressed the opinion that such hemorrhages are probably more usual in this disease than is generally supposed and are not discovered simply because they are not looked for; but this opinion does not rest upon the secure foundation of an examination of a large number of cases, and until such examinations have been made nothing more definite can be said in regard to the frequency of these extravasations. Indeed, no less an authority than Gowers[6] regards intestinal hemorrhages as an important aid in making a differential diagnosis between septicemia and typhoid fever when the general symptoms do not sharply separate the one from the other; that is, he would expect to find them in the septicemia and *not* in the typhoid fever. A perverted quality of the blood, a weakened condition of the bloodvessel walls (Bull), or a microbic invasion of the vascular coats (Keen) may explain these hemorrhages. W. K. Rogers has described a retinal lesion after typhoid fever, consisting of reddish-yellow clustered spots in the muscular regions, to which he gives the name of excedative retinitis. At least two instances of obstruction of the lumen of the central artery of the retina by an embolus, or perhaps a thrombus, during convalescence trom typhoid fever have been reported (Galezowski, Snell), and one of partial thrombosis of the central retinal vein (W. K. Rogers). Blindness has occurred from a hemorrhage into the optic nerve itself, or into its sheath (Leber, Deutschmann).

Anesthesia of the retina has been described by Leber, and large irregular scotomas and annular defects in the

visual field which have been attributed to alterations in the blood-streams have been recorded by several observers. An *amblyopia without ophthalmoscopic change*, something like that which occurs during scarlet fever, or as the result of a profound toxemia, for example, by lead, has been reported by Ebert, Nothnagel, and others. According to Gowers, it presents a favorable prognosis and disappears in the course of a week or two. Temporary blindness of this character can be caused by the action of toxins circulating in the retinal blood-streams upon the inner retinal layers.

Optic neuritis occasionally occurs during the course of typhoid fever, one variety being unconnected with intracranial complications. In other words, there may be a neuritis of the optic nerve, exactly as there may be neuritis of nerves elsewhere in the body. Another variety develops when this fever is complicated with meningitis, which of itself is a rare complication (sixteen times in seventeen hundred cases collected by Westcott for Keen). A third variety, described by Oglesby as subacute in character, and which he thinks is always associated with symptoms of meningitis, is more frequent among childbearing women than among men. It should be remembered, however, that cerebral manifestations during typhoid fever do not necessarily mean meningitis. Finally, retrobulbar optic neuritis may develop.

Optic nerve atrophy, a post-typhoidal phenomenon, may result from an antecedent neuritis, or from excessive hemorrhage (intestinal, nasal, menstrual), or it may be caused by the injudicious use of quinin. Inasmuch as recent investigations indicate that certain atrophies of the optic nerve which at one time were regarded as primary, are really secondary to degeneration of the ganglion-cell layer of the retina, it is possible that some of the post-typhoidal atrophies depend for their origin upon a profound toxic influence exerted upon the inner strata of the cerebral layer of the retina.

Ward Holden has experimentally shown that excessive hemorrhage in all probability acts in this way in producing optic nerve atrophy.

Affections of the *intraocular and extraocular muscles* occasionally complicate typhoid fever. Thus, during

the period of convalescence dilation of the pupil and paresis of accommodation may be present. Segal describes mydriasis without cycloplegia and with normal vision, which he attributes to irritation of the sympathetic.

Paralysis of the extraocular muscles is an uncommon phenomenon, and probably rarely occurs at the height of the fever in the absence of intracranial complications. Ptosis and abducens palsy have been recorded in the third week of the disease (Nothnagel). During convalescence, according to Knies, extraocular palsies are more frequent, certain varieties appearing quickly and quickly relapsing, which this author believes to be nuclear in origin, and which he attributes to a chronic nephritis, a frequent sequel, in his opinion, of typhoid fever. Complete oculomotor palsy, however, as I have shown,[7] can occur at this stage without such an etiology. Certain muscular paralyses which have been noted at long intervals after typhoid fever have been attributed by their reporters, for example, Runeberg, who noted trochlear paralysis one and one-half years after the fever, to the typhoid poison. They are difficult to understand and their association with the disease is not at all proven. During mild attacks of typhoid fever in children there may be strabismus and retraction of the head, which disappear in a few days. Perhaps these symptoms are due to a basilar meningitis, but they may also be explained by an intense effect of the poison on the nervous system without the production of inflammation. In a case of oculomotor palsy, such as I have reported, if meningitis is set aside as the etiologic factor, we may attribute it to an action of the poison on the third nerve precisely as it acts on other nerves, usually those of the arms and legs.

Among the *affections of the orbit* and of the orbital circulation which have occurred during the course of typhoid fever, and which must be exceedingly rare, the following may be mentioned : Thrombosis of the orbital veins; orbital cellulitis, in one instance a culture from the pus developing the presence of the bacillus of Eberth (Panas); and spontaneous orbital and intraocular hemorrhage. The last-named affection has been described by C. A. Findlay in the third week of

typhoid fever and was ascribed to a degenerative change in the walls of the bloodvessels in connection with the general lowering of nutrition. Purpuric spots appeared on the patient's body.

Finally, a word may be said with reference to the relation of typhoid fever to *asthenopia*. Patients frequently say, "I never experienced any trouble with my eyes until after I had typhoid fever," and then wish to know whether the typhoid fever has in some way altered eyes which previously were supposed to be perfect. Typhoid fever may materially lessen accommodative power, as has already been pointed out; hence injudicious reading during protracted convalescence may cause asthenopia. Again, as Landolt has shown, this fever is a fruitful source of weakness of the adductive power which then remains to plague the patient, "the vestige of a debilitating disease," as he expresses it. But in a certain number of patients the asthenopia appears to be most marked when the early stage of convalescence has passed and strength has been regained, often with marked increase in the body-weight. Perhaps these cases may be explained by what Gould calls the law of refraction-change following increase or decrease of the body-weight. He believes that great increase of the body-weight may cause shrinking of the antero-posterior diameter of the eyeball, or alteration of curvature; that is, increase of hyperopia, decrease of myopia, or similar changes of astigmatism. It is easy to infer from all this that a thorough examination of accommodative power, muscular balance, and refractive error after recovery from typhoid fever is indicated should symptoms referable to the eyes arise.

REFERENCES.

[1] Die Beziehungen der Sehor. ans und seiner Erkrankungen zu den übrigen Krankheiten des Körpers und seiner Organe, Wiesbaden, 1893.
[2] *Medical Record*, New York, 1897, Vol. li, p. 577.
[3] *Medical News*, New York, Vol. lxxv, 1899, p. 614.
[4] Doubtless feebleness of accommodative power as part of a general post-febrile weakness is the most frequent ocular complication.
[5] Keen: Surgical Complications and Sequels of Typhoid Fever, p. 304.
[6] *Trans. Amer. Oph.*, Sec. viii, 1899, p. 535.
[7] Medical Ophthalmoscopy, Third Edition, 1890, p. 297.
[8] *Journal of Nervous and Mental Diseases*, 1899.